ASSESSMENT OF THE RISK FOR SILICOSIS AND LUNG CANCER ASSOCIATED WITH RESIDENTIAL EXPOSURE TO RESPIRABLE CRYSTALLINE SILICA ORIGINATING FROM THE "POINT OF THE MOUNTAIN" GRAVEL QUARRY

Environmental Epidemiology Program
Bureau of Epidemiology
Utah Department of Health
PO Box 142104
Salt Lake City, Utah 84114-2104

Updated: December 4, 2018

Roles, Responsibilities, and Capacity of the Environmental Epidemiology Program
The Environmental Epidemiology Program (EEP) has three primary roles:

- Provide a public resource of health data and monitoring data associated with environmental health concerns;
- Provide assessment and interpretation of data to characterize potential environmental risks, and recommend actions to monitor and mitigate those risks;
- Develop and maintain administrative rules on injury surveillance and public health sanitation standards.

To meet these responsibilities, the EEP staff includes expertise in epidemiology, toxicology, risk assessment, risk communication, biostatistics, geomatics, health education and environmental health sciences.

The EEP does not have the role, responsibility or capacity to:

- Conduct permitting or regulating activities, or enforce rules;
- Conduct sampling and testing of soil, water, air, or homes for environmental contaminates (except at a very limited level).

Defining a Public Health Concern

The Centers for Disease Control and Prevention (CDC) has adopted the Vilnius-Dandoy criteria (CDC 2013; Vilnius & Dandoy 1990) for evaluating and establishing public health priorities. The Vilnius-Dandoy criteria were researched and established at the request of the Utah Department of Health (UDOH). Dr. Dandoy served as the Executive Director of the UDOH at the time. Those criteria are as follows:

- **Size of the risk**:
  Epidemiologic Measurement of Risk: Epidemiologists measure risk as a comparative ratio of disease burden measures (for example, the rate of disease) between two populations. There are a number of types of risk ratios including: odds ratios (OR), relative risks (RR), standardized incidence ratios (SIR), or standardized mortality ratios (SMR). The odds ratio (a measure of the probable association of a risk factor to a health outcome) is a commonly used measurement for exposure risk assessment. All of the risk

measures are comparative ratios, comparing a study group to a “comparison” group. The study group might be a population exposed to respirable crystalline silica (rc-silica). The comparison population could be one that is not exposed to rc-silica. If the disease burdens of the two groups are equal, the risk ratio will be 1.0. If the study group (the exposed group) has a higher disease burden compared with the comparison group, the risk ratio will be greater than 1.0. If this comparison is in the form of an odds ratio, then the exposure risk is considered to be “associated” with the health outcome. Good epidemiology is seldom able to declare that a risk factor is “causal” without the assistance of other disciplines such as toxicology (Greenland et al. 2008; Griffith et al. 1993).

Because there is a lot of random variation in the association of risk factors to health outcomes (consider, for example, the variation in the association between tobacco use and lung cancer) a “95% confidence interval” (95% CI) is applied to the risk ratio. Occasionally another percentage is used, such as 99%. The real interpretation is actually the lower limit. If the lower limit of 95% CI is greater than 1.0, then the risk is considered “significantly associated.” If the lower limit of the 95% CI is less than 1.0, then we generally say there is no significant association. Some epidemiologists may say there is a weak association (Greenland et al. 2008; Griffith et al. 1993).

One way to interpret a risk ratio is as a magnitude of excess risk. A risk ratio of 1.0 is interpreted as a no excess risk. A risk ratio of 2.0 can be interpreted to mean the study population has twice as much risk as the comparison population. Conversely, a risk ratio of 0.5 can be interpreted to mean the study population has half the risk as the comparison population.

Meaningful Risk Standard: The CDC recommends a “meaningful” criterion to the size of the risk. The EEP applies this guideline. Much of environmental epidemiology is driven by cancer concerns, so the recommendations used by environmental epidemiology often come from cancer investigations which are then applied to other types of investigations.

In the 1990’s, a risk ratio needed to be in the 50s to be considered meaningful (Bender et al. 1990). Fortunately, more government public health agencies have since relaxed the meaningful criteria. Currently, the EEP uses the recommendation of Thun and Sinks (2004) which considers a risk ratio above 3.0 to be meaningful. These criteria are open to change as the literature evolves and as directives to the EEP are issued.

Toxicological Measurement of Risk: Toxicologists measure risk as a comparison of effect levels and effects (dose-response). Toxicological investigation of effect levels attempts to determine the dose where an effect starts or stops manifesting. These levels are known as the “lowest observable effect level” (LOEL) or the “no observable effect level” (NOEL). If there are both positive and negative effects associated with an exposure, toxicologists will distinguish a “lowest/no observable *adverse* effect level” (LOAEL or NOAEL, respectively).

From these dose-response levels, and considering the potency, exposure routes, frequency, and the susceptibility of the exposed individual, toxicologists develop an exposure reference dose (RfD). Typically, the RfD is the NOEL or NOAEL divided by several orders of magnitude (i.e., 10, 100, 1000, etc.) to account for uncertainty. The degree of uncertainty is based on the nature of the studies used to find the dose-response relationship. For example, was the finding a NOEL or a LOEL? How close to humans was the animal model used – human subjects, apes, rabbits, mice, etc.? Additional uncertainty is added because of the variability in humans in their response to exposures. Thus, if the NOAEL for a respiratory exposure to a chemical was found to be 1.0 mg/m^3 (read 1.0 milligrams of chemical per cubic meter of air), the RfD might be set at 0.001 mg/m^3, or a thousand times below the known “no observable adverse effect level” (Faustman & Omenn 1996).

Toxicologic Risk Level: The US Occupational Health and Safety Administration (OSHA) has established many 8-hour time-weighted-average (TWA) permissible exposure limits (PELs) for contaminants in the workplace. The OSHA TWA PEL for rc-silica is 50 $\mu g/m^3$ (read 50 micrograms silica per cubic meter of air). OSHA has also established an action level of 25 $\mu g/m^3$ (OSHA 2017). Air levels at or above the action level require that the work site start monitoring and mitigating the air exposure. Air levels above the PEL result in a regulatory action (i.e., a fine, an order to mitigate, etc.).

The US Environmental Protection Agency (EPA) data and guidelines suggest the dose-response for a continuous (24-hour) exposure of 8 $\mu g/m^3$ would pose about the same risk in the general public as an 8-hour exposure of 22.4 $\mu g/m^3$ does in an occupational setting (EPA 1996). While this research did not establish a federal standard for non-occupational exposure to respirable silica, it does suggest that the OSHA TWA PEL can be interpolated to 18 $\mu g/m^3$ (50 $\mu g/m^3$ x 8 $\mu g/m^3$ / 22.4 $\mu g/m^3$ = 17.9 $\mu g/m^3$) averaged over a 24-hour period.

In 2005, the California Office of Environmental Health Hazards set a chronic reference exposure level (cREL) for $PM_{4.0}$ at 3.0 $\mu g/m^3$ (Richards et al. 2009). While the EEP uses federally established standards or recommendations to interpret public health risk, we do consider what other states are using.

- **Seriousness of the outcome**: This criterion has two factors – urgency and severity. All of the health outcomes (silicosis, chronic obstructive pulmonary disease [COPD], lung cancer, gastrointestinal cancer, kidney damage, autoimmune disease [particularly rheumatoid arthritis], increased risk for contracting tuberculosis [if exposed], etc.) associated with rc-silica exposure are severe (ATSDR 2017; Bernstein et al. 2013). All of these outcomes have long latent periods of 5–30+ years (i.e., the time between an exposure and the onset of a disease or symptoms). Since 1990, the residents of Traverse

Mountain (or the city of Lehi when we do not have the ability to resolve the data to Traverse Mountain) have experienced the following disease burdens:

Health Outcome	Data Source	Resolution	Rate per 10,000 population/year	Standardized Incidence Ratio (95% CI)
Silicosis	Hospital Discharge Database	City of Lehi	0.00	NA
COPD	Hospital Discharge Database	City of Lehi	5.6	0.43 (0.34 – 0.55)
Lung & Bronchial Cancers	Utah Cancer Registry	Traverse Mountain	0.2	0.11 (~0.0 – 0.61)
Gastrointestinal Cancers (stomach, small and large intestines, & rectum)	Utah Cancer Registry	Traverse Mountain	1.4	0.41 (0.15 – 0.90)

The comparison population is the rest of the state of Utah. If there is a sudden dramatic change in the rates or risk ratio, that could indicate an emerging public health concern. They are also useful in educating the public about the health status in the neighborhood.

In this case, for interpretation, there have been too few cases of silicosis in the city of Lehi to make a statistical evaluation. The rate of COPD in the city of Lehi is about half what the rest of the state is experiencing, and that difference ***is*** statistically significant. However, the only data available to the EEP are those cases admitted for treatment at a hospital. The rate is likely underestimated. If the assumption that the ratio of hospitalized cases versus outpatient cases is approximately equal for both the city of Lehi and the state of Utah, then the SIR will remain an accurate assessment of the risk difference between the city Lehi and the State.

Similarly, the cancer incidence rates among Traverse Mountain residents are significantly lower than the rest of the state (the upper limit of the 95% CI is below 1.0). The cancer data currently available to the EEP is through 2015. We have just received and are processing 2016 data. The number of cases were too small to evaluate trends. Most of the growth in Traverse Mountain appears to have occurred after 2001 and all of the cases are scattered through the 2001–2015 time frame. For reference, the EEP's cancer statistical review procedures are found at:

health.utah.gov/enviroepi/cancerstudies/Documents/Utah_Cancer_Investigatio_Protocol_Final_2May2016.pdf

The data presented in the table are incidence rates—meaning that the residential address at the time of initial diagnosis was in Traverse Mountain. We assume the prevalence rates (which counts cases diagnosed in Traverse Mountain residents and cases diagnosed elsewhere and moved in) would be higher. Both lung cancer and GI tract cancer have long latencies, typically in the 15–30+ year range (Nalder & Zurbenko 2014; Thun et al. 2018). We think the triggering events (the combination of genetics, life choices, environmental exposures, infections, medical treatments, and random chance) (Thun et al. 2018) occurred prior to residency in Traverse Mountain for most, if not all, incident cases.

Incidentally, Traverse Mountain residents have a non-significant elevated rate of skin cancers (SIR=1.13; 95% CI = 0.18-3.57), breast cancer (1.31; 0.33-3.41), prostate cancer (1.72; 0.43-4.49), and thyroid cancer (1.46; 0.23-4.60) during the last five years (2011-2015) of data.

- **Political will to address the problem**: This criterion has two components—public concern and availability of resources. This is an area where citizen activism has the most value. The governor or the state legislature (through the enactment of law) can direct the UDOH or the EEP to designate an issue as a public health concern. For example, initially, the cancer rates in Monticello did not meet the criteria for a concern. A citizen's group successfully influenced the governor's office to make the Monticello cancer rates a public health concern. In the end, the city of Monticello, with the assistance of the EEP, were able to obtain federal grant money to increase their local capacity to screen for cancers. In another example, the EEP evaluated the risk of a type of chicken feed containing arsenic to be a public health problem. The manufacturer lobbied state leadership which then directed the EEP to retract our report about the concern. In both cases, interested parties used the political system to establish a political will the EEP addressed.

- **An intervention**: The EEP does not have the authority to shut down rock quarry operations, nor does it have the authority to make recommendations to shut it down. That authority lies with the Utah Department of Natural Resources (UDNR) and the Utah Department of Environmental Quality (UDEQ). The kinds of interventions the EEP can recommend would focus on residents themselves, and protective actions they can take. For example, the EEP has recommended that all residents:
    - Avoid smoking or using tobacco products
    - Minimize alcohol consumption
    - Participate regularly in physical exercise or deep breathing exercises
    - Help prevent respiratory infections through regular hand washing, good oral hygiene, and staying current on vaccinations
    - Get regular healthcare checkups

- Minimize exposure to air pollution as much as possible
    - Stay indoors if no protective equipment (i.e., an effective respirator mask) is available
    - Use protective equipment if outdoor activity is necessary
    - Choose less strenuous activities when air pollution levels are high

  We believe these recommendations reduce the risk of developing silicosis and subsequently developing lung cancer.

- **Health disparities**: The EEP's practice is to reduce the threshold for meaningfulness if the exposed population is socially or economically deprived. The EEP does this because these populations typically have less access to health care and ability to manage their concerns privately.

Understanding the Risk: Approach 1 – Literature Review

The EEP was asked by the Lehi City Council to address rc-silica. The EEP has reviewed a number of other gravel and sand quarries in the state of Utah in the past, including the one in Box Elder County (ATSDR 2006).

The EEP has not addressed, nor been asked to address, other potential air pollution hazards associated with the Point of the Mountain.

- **Respirable crystalline silica (rc-silica)**: rc-silica < 10 µm (micrometers) in size are able to penetrate to the alveolar sacs of the lungs (ATSDR 2017; Bernstein et al. 2013; Pinkerton & Southard 2005). Deposition of rc-silica occurs by two mechanisms. Approximately 60% of particles < 0.1 µm in size deposit in the alveolae by diffusion. Approximately 60% of particles between 1.0 and 2.5 µm deposit in the alveolae by impaction. Particles in the range of 0.1 to 1.0 are not able to deposit by either mechanism and are predominately exhaled. Particles larger than 2.5 µm deposit higher up (mouth and nasal cavity, pharyngeal area, trachea, the bronchi, and the bronchioles) in the respiratory tract (Bezemer & Pieters 2009; Brown 2015; Frohlich et al. 2016; Heyder 2004; Hussain et al. 2011; Lin et al. 2009; Namati et al. 2008; Rissler et al. 2017; Shulz et al. 2000).

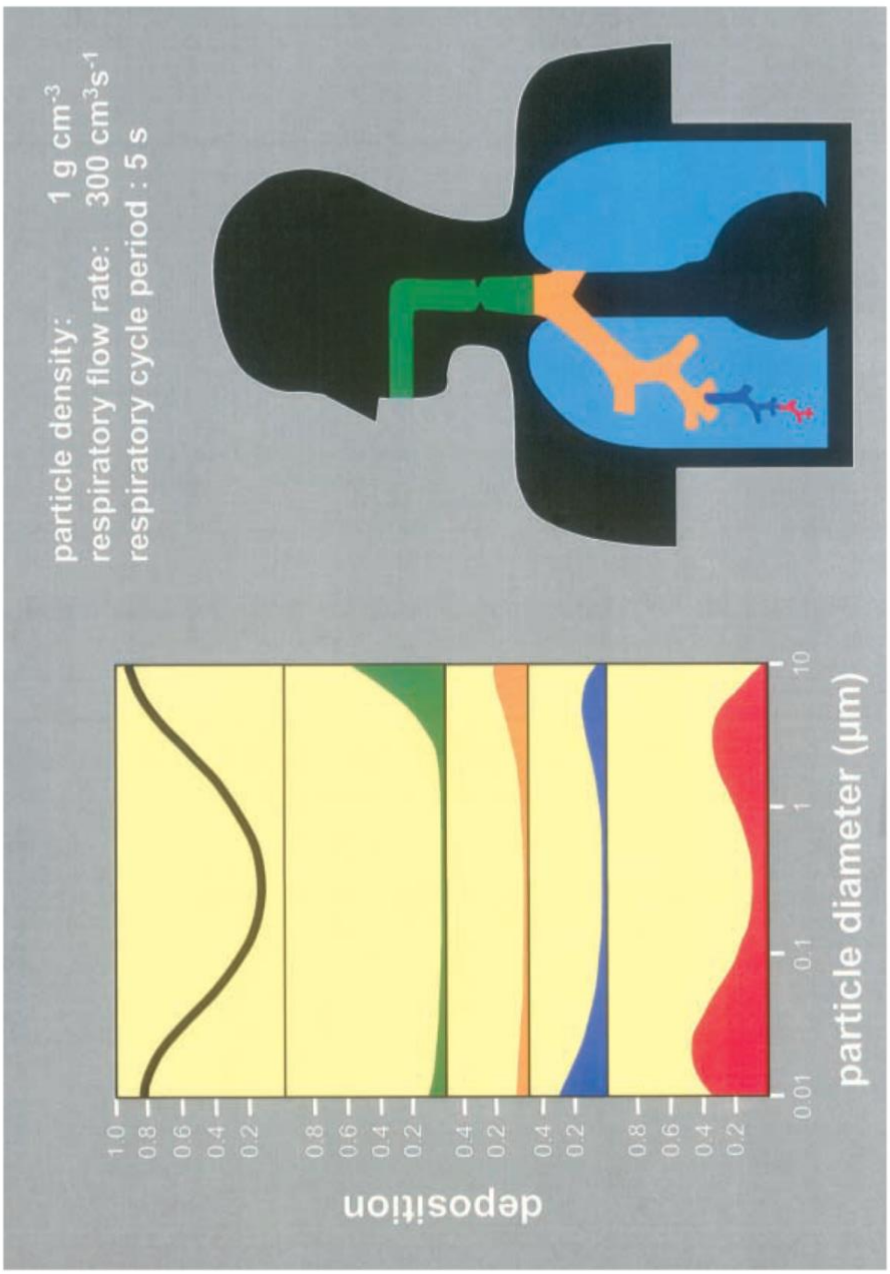

Figure 2. Total and regional deposition of unit-density spheres in the human respiratory tract predicted by the ICRP deposition model for oral inhalation at rest.

The red graphic represents the alveolar deposition. Note the x-axis is logarithmic. (*figure from Heyder 2004*)

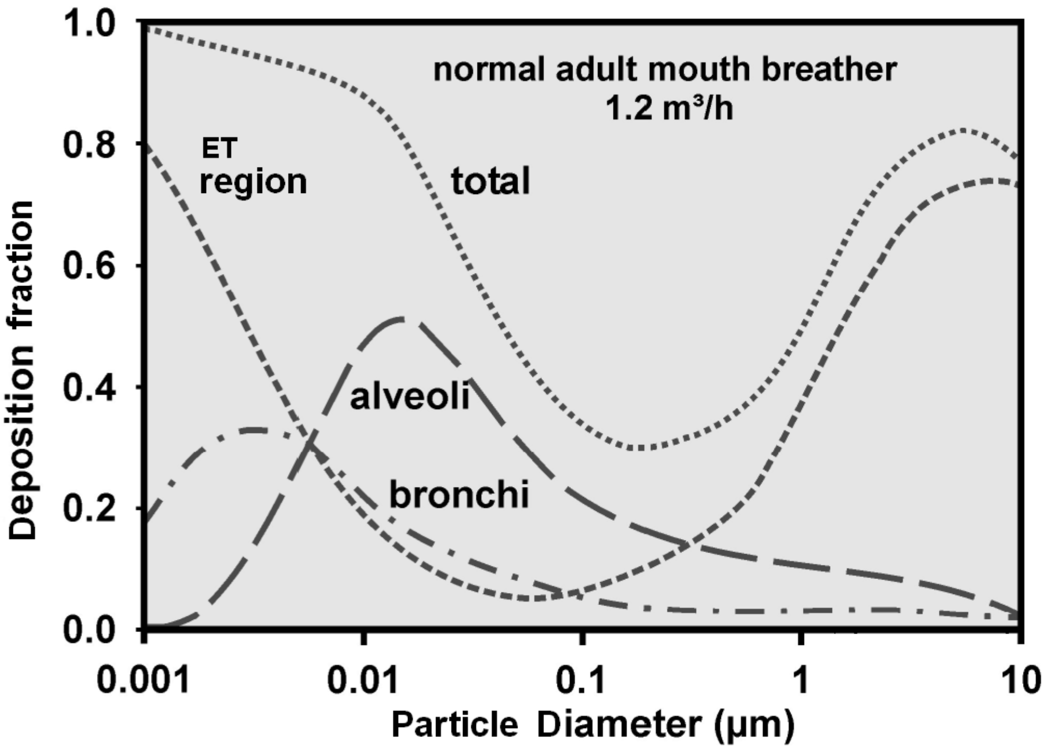

Figure 6: Average predicted total and regional lung deposition based on ICRP [1] deposition model for nose breathing for light exercise breathing condition. Highest deposition (ET region for 0.001 and 10 μm particles, bronchi region for 0.005 to 0.007 μm particles and alveolar region for 0.01 to 0.05 μm particles).

Note x-axis is logarithmic. (*figure from Hussain et al. 2011*)

rc-Silica is poorly soluble. Deposition results in irritation that initiates a number of pulmonary clearance processes. If the level of rc-silica is high enough to overload the clearance processes, then encapsulating processes are initiated. The encapsulating processes result in the development of the fibrotic condition known as silicosis (Bevan et al. 2018).

- **Silicosis**: Silicosis is a progressive, irreversible lung disease. Only rc-silica that is able to penetrate the alveolar sacs causes silicosis. Amorphous silica does not cause silicosis. rc-Silica that is too large to penetrate to the alveolar sacs does not cause silicosis (ATSDR 2017; Bernstein et al. 2013; Thomas & Kelley 2010; Yang et al. 2006).

Chronic silicosis is a progressive disease that presents first as simple silicosis (also known as Stage I silicosis), characterized by tiny round scar lumps developing in the alveolar dense regions of the lungs. Simple silicosis progresses to progressive massive fibrotic silicosis (PMF or Stage II), characterized by large fibrotic masses that develop in the lung tissues. PMF silicosis may progress to PMF Stage III silicosis characterized by *cor pulmonale* (abnormal enlargement of the heart). All forms of chronic silicosis are characterized by stressed breathing. The average latency period for developing silicosis is 22.9 years after the start of exposure in the occupational setting (persistent unprotected exposure lasting up to 8-hours a day). Approximately 48% of cases of simple silicosis progress to PMF (Steenland & Ward 2014; Yang et al. 2006).

The EEP uses several health-based searchable literature search engines such as PubMed and ToxNet. These search engines provide an index of peer-reviewed scholarly articles. A search of PubMed for "silicosis" listed 6,024 articles 1923 and through the most recent publication in November 2018. Of those, a little over 180 articles address risk magnitude associated with rc-silica exposure. The EEP reviewed those articles.

The exposure-response relationship of rc-silica to chronic silicosis is non-linear (Calvert et al. 2003; EPA 1996; Mannetje et al. 2002).

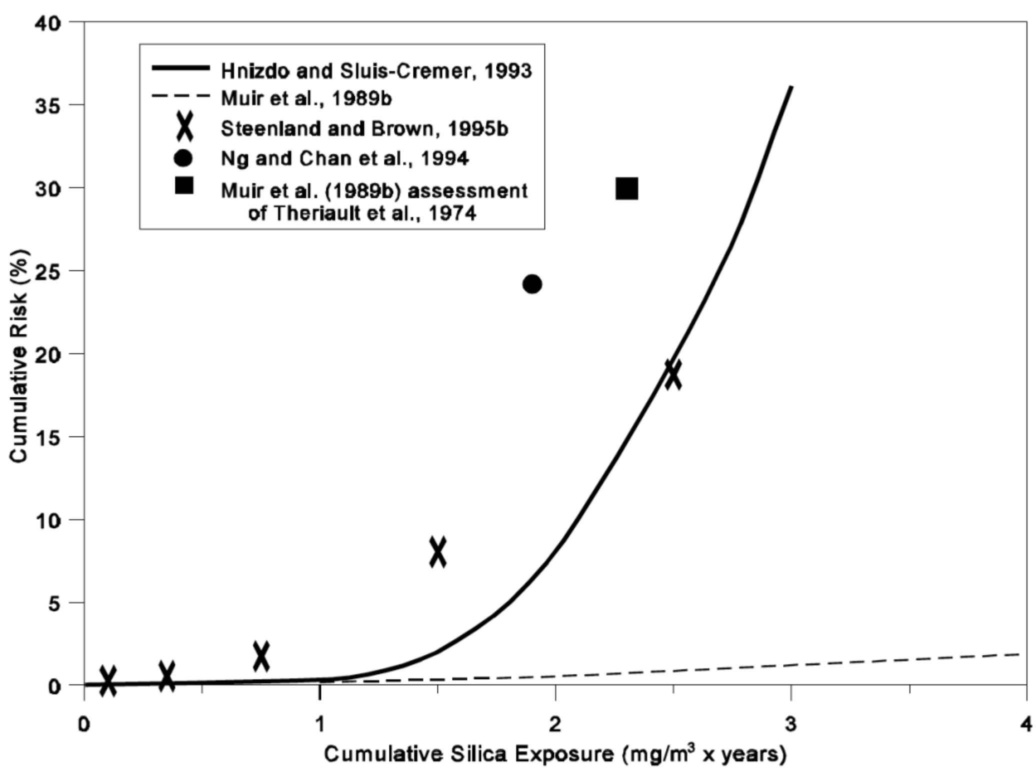

Figure 7-1. Cumulative silica risk curves estimated for South African gold miners (—) (Hnizdo and Sluis-Cremer, 1993) and Canadian hardrock miners (– –)(Muir et al., 1989b); cumulative silica risk points estimated for South Dakota gold miners (✗) (Steenland and Brown, 1995b), Hong Kong granite workers (●) (Ng and Chan, 1994), and Vermont granite miners (■) (Theriault et al., 1974c; Muir et al., 1989b).

(*figure from EPA 1996)*

We interpret the figure above to suggest that exposures below 1 mg/m^3-years (read as 1 mg exposure per cubic meter of air per year) pose minimal risk. A 1 mg/m^3-year exposure is equivalent to 1,000 µg/m^3-years (read as 1,000 micro grams exposure per cubic meter of air per year). Risk begins to rise from 1.0 to 1.5 mg/m^3-years (1,000-1,500 µg/m^3-years), and greater than 1.5 mg/m^3-years (>1,500 µg/m^3-years) the risk increases substantially. These studies are for a 30-year cumulative exposure.

Calvert et al. (2003) found that the risk of developing silicosis to be OR = 2.9 (2.4 – 3.5) when exposed to the OSHA TWA PEL (50 µg/m^3 over 8 hours; read as exposure to an average of 50 µg per cubic meter of air for 8 hours). The odds ratio increased to 6.8 (5.7 – 8.2) when the exposure was twice the PEL (100 µg/m^3) and to 30.5 (18.4 – 50.5) when the exposure was five times the PEL (250 µg/m^3).

Similarly, a persistent occupational exposure (8 hours/day) lasting 30 years to 50 µg/m^3 results in a less than 5% increased lifetime risk for silicosis, whereas, a 30-year persistent occupational exposure of 100 µg/m^3 results in a 25% increased lifetime risk for silicosis (Finkelstein 2000; Mannetje 2002).

Vacek et al. (2018) reports an increasing trend in the risk as cumulative exposure increases. The study assessed a cohort of 1,902 workers from 40 different work sites in 22 different states. Workers with cumulative exposures between 500 and 1,500 µg/m^3-years had an odd ratio of developing silicosis of OR = 1.10 (0.26 – 4.64). For exposures between 1,500 and 3,000 µg/m^3-years, the OR = 2.06 (0.55 – 7.79). For exposures greater than 3,000 µg/m^3-year, the OR = 12.57 (2.08-55.47).

Chronic silicosis has been associated with the development of COPD (chronic bronchitis and emphysema are examples of COPD), lung cancer, gastrointestinal cancer, kidney damage, autoimmune disease, and an increased risk for contracting active tuberculosis if exposed to the bacteria (for example, rheumatoid arthritis) (ATSDR 2017; Bernstein et al. 2013).

Exposures at extreme levels may result in acute silicosis or accelerated silicosis which are usually fatal due to anoxia a few months to years after exposure (ATSDR 2017; Bernstein et al. 2013; Yang et al. 2006).

- **Lung cancer from rc-silica exposure**: The only health outcome the EEP was asked to address was the development of lung cancer from rc-silica exposure.

  Nature of Public Research: Unlike some of the more exact sciences, epidemiologic research into the association of a specific risk factor to a specific health outcome is subject to high variability. For example, despite the evidence that rc-silica is associated with the development of silicosis, Checkoway et al. (1997) studied a large occupational cohort (2,342 workers) and found no significant risk for cancer deaths (SMR = 1.1; 0.9 – 1.2) and a low significant risk for deaths from respiratory disease (1.8; 1.4 – 2.2).

A meta-analysis found the reported relationship between silicosis and lung cancer to range from statistically significant and meaningful (SMR = 6.03; 4.38 – 8.09) to statistically insignificant (1.37; 0.95 – 1.91). In that analysis of 31 individual studies, seven (23%) had insignificant findings. Twenty-four (77%) showed a significant association of silicosis exposure to lung cancer, but only two (6%) resulted in meaningful findings by the EEP's current definition (Lacasse et al. 2005).

Similarly, the meta-analysis by Poinen-Rughooputh et al. (2016) included studies that ranged from 0.90 (0.51 – 1.47) to 6.03 (5.29 – 6.77). In that report, 23% of the included studies were statistically insignificant and 9% were of a magnitude to meet the EEP's "meaningfulness" threshold.

Because of this variability, the EEP preferentially (but not exclusively) uses meta-analytical reports. Meta-analysis is a statistical process of summarizing the wide-ranging body of literature into a single assessment. Using meta-analytical reports has several advantages, in that it ensures the interpretation of the assessment has the statistical power of the combined reports, and ensures that an incomplete literature review is not guilty of "cherry-picking" reports to support a predetermined conclusion (Hedges & Olkin 1985).

Non-Occupational Exposure: Unfortunately, there is a scarcity of studies on non-occupational exposures. Here, reports on occupational exposure are considered and, where possible, extrapolated to non-occupational exposures.

Meta-analysis of the association of rc-silica exposure to the development of lung cancer: The following meta-analytic reports are from the occupational setting.

	Erren et al. 2011	Poinen-Rughooputh et al. 2016	Smith et al. 1995
Number of studies	38	10	29
Lung cancer risk among silicotic cases	2.1 (2.0 – 2.3)	2.3 (1.9 – 2.8)	2.2 (2.1 – 2.4)
Lung cancer risk in non-silicotic cases not adjustments	1.2 (1.0 – 1.4)	1.2 (0.9 – 1.6)	
Lung cancer risk in non-silicotic cases adjusting for smoking	1.0 (0.8 – 1.3)		

All of the above ratios are relative risks (RR) with 95% confidence intervals.

The three meta-analyses presented here may have some overlap in which individual studies they included. They are consistent in their findings that lung cancer development is about twice as high in silicotic cases but not significant in non-silicotic cases. In other words, the development of silicosis after rc-silica exposure in the occupational setting is associated with the development of lung cancer. However, rc-silica exposure is not directly associated with lung cancer development.

Steenland et al. (2001; 2014) in a meta-analytic report that examined 10 studies suggested that a lifetime occupational exposure to 0.10 mg/m^3 (100 µg/m^3) of rc-silica results in increased lifetime lung cancer risk of 1.1% to 1.7% above background risk.

The EEP replicated meta-analysis using 181 reports of risk magnitude and found similar pooled results. Each of the authors cited above, and EEP noted (using the funnel diagram) in its own analysis that there is a significant publication bias associated with these studies. Publication bias is effect caused when researchers and publishers do not publish insignificant or negative findings, thus biasing the pooled effect towards a positive finding.

Meta-analysis of the association of rc-silica exposure and death by lung cancer: These meta-analytic reports are in the occupational setting.

	Lacasse et al. 2005	Poinen-Rughooputh et al. 2016	Smith et al. 1995
Number of studies	31	85	29
Lung cancer deaths among silicotic cases without adjustment	2.5 (1.6 – 3.7)	2.3 (1.9 – 2.8)	2.7 (2.3 – 3.2)
Lung cancer deaths among silicotic cases adjusting for smoking	1.6 (1.3 – 1.9)		
Lung cancer deaths among in non-silicotic cases without adjustment		1.8 (1.1 – 3.0)	

All of the above ratios are standardized mortality ratios (SMR) with 95% confidence intervals.

The three meta-analyses presented here may have some overlap in which individual studies they included. They are consistent in their findings that lung cancer deaths occur two to three times more often among silicotic cases (not adjusted for smoking).

Calvert et al. (2003) found that the risk for developing lung cancer from occupational silicosis was OR = 0.88 (0.87 – 0.90) when exposed at the OSHA TWA PEL level. This risk increased to 1.1 (1.1 – 1.2) for exposures twice as high, but did not change for exposures five times as high.

The data suggest that silica exposure is a risk effect modifier of tobacco use the development of silicosis, COPD and lung cancer. However, the data is limited (Hessel et al. 2003, Tse et al 2014). An effect modifier is a factor that enhances the primary effect of another risk factor. In this case, tobacco use is the primary risk. Silica exposure increases the harm done by tobacco use.

- **Other findings**:
  - Two studies found that the number of aerosol particles that reached the lower pulmonary regions in children was the same or lower than in adults. This finding is attributed to the lower tidal volume and inhalation pressure of children, allowing the particles to impact in the extra-thoracic part of the respiratory tract (Albuquerque-Silva 2014; Rissler et al. 2017). Our assessment is that both studies are a start, but too weak (3 and 7 subjects, respectively) to be definitive.
  - In addition to basic comparative measures of risk (odds ratios, relative risk, etc.), epidemiologists have a number of useful tools to measure the attribution of risk to various factors when a combination of risk factors are present. For example, the risk factors for lung cancer include tobacco exposure, radon exposure, exposure to heavy metals (e.g., arsenic, beryllium, cadmium, etc.), exposure to organic compounds (i.e., vinyl chloride), certain dietary supplements, genetics, combustion engine exhaust, radiation therapy, and a number of other factors (CDC 2018). The basic measures of risk are problematic in that they conceal the background or confounding risks (Suissa 1999). A large cohort study (58,677 workers) estimated that the excess relative risk (ERR) per dust-year associated with silicosis was 0.061 (95% CI = 0.039 – 0.083) for exposures > 10 mg/m^3-year (10,000 $\mu g/m^3$-year). This study confirms a small positive exposure-response relationship between silica and lung cancer for very high exposures (Sogl et al. 2012).
  - Individuals with genetic variations in certain genes (the tumor necrosis factor (TNF-alpha), and interleukin cytokine (IL-10) genes) have increased risk of developing silicosis after rc-silica exposure (Kurniawidjaja 2014).

- **Research by Utah Physicians for a Healthy Environment (UPHE)**: As we have previously stated, we applaud the work done by UPHE. They have been able to address concerns much more broadly than the EEP has been authorized to do. We encourage policymakers to consider their work in parallel with ours.

Understanding the Risk: Approach 2 – Air Dispersion Modeling

- **Point of the Mountain**: The rock formations in the quarries at Steep Mountain, Sage Canyon, and other nearby areas (collectively referred to as "the Point of the Mountain") are highly fractured orthoquartzite with some limestone and calcareous sandstone. Quartzite is approximately 90% quartz crystal (cristobalite or tridymite), which is in turn predominately crystalline silica (c-silica) (JBR 2009).

Geneva Rock is the largest of a number of companies operating at the Point of the Mountain and is permitted to emit up to 128.86 tons of PM_{10} per year (read as particulate matter smaller than or equal to 10 microns in size per year) (JRB 2009). There was no mention of a permitted level for total solid particulate matter (TSP). It is quite possible that the air opacity is affected by a range of air buoyant particle sizes up to about 500 microns.

In addition to rock quarry operations, the Point of the Mountain site includes concrete batch production, hot mix asphalt production, as well as fuel farms and maintenance operations.

Using inhouse geographic information systems (GIS) and the most recent satellite imagery, the site includes approximately 2,939,910 square meters of de-vegetated or excavated land.

- **Sand and gravel sources of silica**: Huggins and Meyers (1986) investigated the size distribution of respirable particles from 29 mines (including at least 15 sand and gravel quarries) and found that about 60% of the particles in the respirable size range (0 to 15 µm) were in the alveolar deposition size range (0 to 3.0 µm). Cauda et al. (2014), investigating mines across the nation, found that, on average, only 9.4 to 11.7% of the total dust cloud mass was rc-silica.

  Richard et al. (2009) found that the PM_4 fraction (read as particulate matter smaller than or equal to 4 microns in size) of the dust from California screening operations, tertiary crushers, and conveyor transfer points comprised 0.000006 to 0.000110 lbs/ton (pounds per ton) of the total dust cloud mass. They further found good correlation between the proportion of rc-silica in the dust cloud mass and the proportion of c-silica in the source material. Using this correlation slope, the EEP estimates that for a source that is 90% orthoquartzite, rc-silica dust would comprise 0.0003 lbs/ton of the total dust cloud mass.

- **Other dust sources**: Within three miles (5,000 meters) of the Traverse Mountain residential area are at least 15 large agriculture fields that are plowed and harvested periodically. Within three miles of the Traverse Mountain residential area, including within the Traverse Mountain community itself, there are a number of housing development areas with active excavation and earth pilling.

  Surveys of crystalline silica levels, while not extensive, suggest that air concentrations between 1 to 10 $\mu g/m^3$ are common in both urban and rural settings and are thought to be from natural sources. Silica is the most common material found on the earth. For this reason, the EPA has put off issuing a reference dose for non-occupational exposures. (Hardy & Weill 1995).

- **Windrose data**: Windrose data indicates that in the Lehi area, about 25% of the year the wind is from the N (3%), NNW (4%), NW (6%), WNW (7%), or W (5%). The Windrose provides an estimation of windspeeds: averaging 1.5 mph for 6% of the year, 5 mph for 12% of the year, 10 mph for 5% of the year, and 15 mph or faster for 1% of the year. The remaining 75% of the year, the wind is either in a direction away from the Traverse Mountain residential area or is calm.

- **Worst case scenario area source exposure modeling**: The EEP used the SCREEN3 air quality dispersion modeling tool to estimate the area-based dispersion of fugitive dust from the quarry site to various distances toward Traverse Mountain.

  The SCREEN3 model gives generic pollutant concentrations at points that are various distances from the source. It is a screening model and is designed to overestimate the pollutant concentration. If the screening results are above a threshold of concern, a more robust model that can account for the meteorological, terrain, and structural conditions is used before making any conclusions (EPA 1995; Bruce et al. 2014; Till & Meyer 1983; Reed 2005; Turner 1994).

  In using this model, the following assumptions were applied (these represent worst case conditions for dust exposure):
  - The sky was always noon-time sunny, no cloud cover, and no precipitation for 100% of the year (the worst-case stability class).
  - The dust cloud mass is 100% rc-silica in the <0.01 μm or the 1.0-2.5 μm size.
  - Any wind speed is able to lift and transport at 100% capacity.
  - The rc-silica is eternally buoyant (no deposition). Deposition is an interesting concern. In reality, as the dust cloud moves downwind the mass is reduced by deposition, but the ratio of rc-silica to total mass increases because the heavier materials will deposit earlier than the lighter materials (Aluko & Noll 2006).

  The annual average exposure levels for the three modeled distances are as follows:

400 feet (122 meters)	Closest resident	9.19 μg/m^3-year
1,660 feet (506 meters)	Closest resident to large pit	9.57 μg/m^3-year
4,900 feet (1,494 meters)	School	8.87 μg/m^3-year

  The overall annual average exposure level across the Traverse Mountain residential area is <10 μg/m^3. This is below the OSHA TWA PEL (50 μg/m^3) and the EPA interpolated equivalent (18 μg/m^3). It is above the California cREL (3.0 μg/m^3).

Next Steps: We have proposed acquiring several portable PM2.5 monitors and conducting spot checks of locations around the perimeter of the site and in the Traverse Mountain residential area (i.e., near the closest homes, near the school, etc.).

- **Investigation of heavy metals**: With a petition to conduct a formal investigation, we could look at this in more depth. As a preliminary assessment, assuming a total dust exposure level of 10 μg/m^3 (0.000010 g/m^3), this is what we might find:

Metal	Weight Volume (μg/g)	Exposure Level (μg/m^3)	ATSDR chronic-duration inhalation MRL (μg/m^3)
Aluminum	1,800	0.0018000	4,500
Arsenic	13.8	0.0000138	2
Barium	82.3	0.0000823	500
Beryllium	0.187	0.0000002	2
Cadmium	0.28	0.0000003	0.01
Chromium	7.24	0.0000072	0.3 (assuming all is Cr-6)
Cobalt	1.23	0.0000012	0.1
Lead	4.34	0.0000004	50
Manganese	96.9	0.0000969	0.3
Molybdenum	0.751	0.0000008	0.4
Strontium	32.4	0.0000324	0.5
Tin	1.1	0.0000011	2,000 (assuming is inorganic)
Titanium	43.0	0.0000430	0.1
Uranium	10.8	0.0000108	0.8
Vanadium	6.96	0.0000070	0.1
Zinc	22.2	0.0000222	1,000

MRL is a Minimal Risk Level, and is interpreted similarly to a RfD.

The metals that are micro-nutrients or were not detected were not evaluated. The above data from a very preliminary assessment. Our preliminary conclusion is that the metal levels from this one sample are not at a high enough concentration to cause concern.

Conclusion: The EEP was asked to address construction dust with consideration of silica, and the risk of silicosis and lung cancer. We have not addressed any other potential pollutant or health outcome.

Based on our literature review, the EEP cannot state that exposure to rc-silica is "safe." Indeed, long-term exposures at or above 1000 μg/m^3-year have an increasing risk approximately twice that of an unexposed community.

However, the literature review, and subsequent modeling of the Point of the Mountain quarry, suggests to us that the exposures to rc-silica originating from the quarry that are experienced by Traverse Mountain residents are transient (25% of the time of the year) in nature, and well below (approximately 100 times below) the levels where observable effects occur. The modeling also suggests that the exposure levels to rc-silica originating from the quarry and experienced by Traverse Mountain residents are below (about one half) the EPA recommended residential exposure interpolation of the OSHA occupational threshold.

Previous investigations: The EEP referenced the "Brigham City Sand and Gravel Pits" (ATSDR 2006) as one example of several of prior investigative experiences. That investigation was conducted as part of a cooperative agreement UDOH had with the Agency for Toxic Substances and Disease Registry (ATSDR) and is therefore published online. We feel there has been some misinterpretation of that report. Similar to this situation, that investigation focused on particulate matter. In that assessment, the EEP found that particulate matter (PM_{10}) ". . . are a nuisance, but do not pose a health concern . . .". Similarly, the data suggested that total solid particulates were ". . . a nuisance and do not pose a health concern." Those reading the report have focused on a recommendation the EEP made because the investigation focused on particulate matter. We recognized then, and continue to recognize now, there are other concerns that were not investigated.

Residents' concerns: We recognize and appreciate that the residents have individual and community level concerns. We encourage your efforts to continue to work within your political jurisdiction to voice those concerns. We feel that the best policy decisions are made when many voices contribute to the process.

Recommendations

The EEP recommends that either the city of Lehi or residents of Traverse Mountain, through the Utah County Health Department, petition the EEP to conduct a more thorough investigation of the site, to include additional potential air pollutants (i.e., VOCs, PAHs, heavy metals, etc.) and to consider more adverse health outcomes (i.e., cancers, cardiovascular disease, other respiratory disease, reproductive health, children's health, etc.).

Additionally, either the city of Lehi or the Utah County Health Department can propose thresholds of concern for the EEP to use that are lower than a risk ratio of 3.0 or the OSHA TWA PEL (or the EPA interpolation of that PEL for residents).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2006. Health consultation: Brigham City sand and gravel pits; Brigham City, Box Elder county, Utah; EPA facility ID UTXCRA07W000. Division of Health Assessment and Consultation: Atlanta, GA.

Agency for Toxic Substances and Disease Registry (ATSDR). 2017. **Draft Toxicological Profile for Silica (*Draft for Public Comment*)**. US Department of Health and Human Services, Public Health Service: Atlanta, GA.

Albuquerque-Silva I, Vecellio L, Durand M, Avet J, Le Pennec D, de Monte M, Montharu J, Diot P, Cottier M, Dubois F, Pourchez J. 2014. Particle deposition in a child respiratory tract model: in vivo regional deposition of fine and ultrafine aerosols in baboons. ***PLOS One*** 9(4):e95456 (8 pages).

Aluko O, Noll KE. 2006. Deposition and suspension of large, airborne particles. ***Aerosol Science and Technology*** 50:503-513.

Bender AP, Williams AN, Johnson RA, Jagger HG. 1990. Appropriate public health response to clusters: the art of being responsibly responsive. ***American Journal of Epidemiology*** 132(S1):S48-S52.

Bernstein D, Dunnigan J, Hesterberg T, Brown R, Legaspi Velasco JA, Barrera R, Hoskins J, Gibbs A. 2013. Health risk of chrysotile revisited. ***Critical Reviews in Toxicology*** 43(2):154-183.

Bevan RJ, Kreiling R, Levy LS, Warheit DB. 2018. Toxicity testing of poorly soluble particles, lung overload and lung cancer. ***Regulatory Toxicology and Pharmacology*** 100:80-91.

Bezemer GFG, Pieters RHH. 2009. Particle deposition and clearance from the respiratory tract. Institute of Risk Assessment Sciences, University of Utrecht: the Netherlands.

Brown JS. 2015. Chapter 27 – Deposition of Particles. In. Parent RA, ed. **Comparative Biology of the Normal Lung, 2nd ed**. Academic Press: Cambridge, MA.

Bruce J, Walton G, Datson H. 2014. Aspects of empirical dust modeling associated quarries. ***Proceedings of the Extractive Geology Conference 2014***.

Calvert GM, Rice FL, Boiano JM, Sheehey JW, Sanderson WT. 2003. Occupational silica exposure and risk of various diseases: an analysis using death certificates from 27 states of the United States. ***Occupational and Environmental Medicine*** 60:112-129.

Cauda E, Joy G, Miller A, Mischler S. 2014 Analysis of the silica percent in airborne respirable mine dust samples from U.S. operations.

Centers for Disease Control and Prevention (CDC). 2013. Prioritizing Public Health Problems Training Module.

Centers for Disease Control and Prevention (CDC). 2018. What are the Risk Factors for Lung Cancer? CDC website. https://www.cdc.gov/cancer/lung/basic_info/risk_factors.htm.

Checkoway H, Heyer NJ, Seixas NS, Welp EAE, Demers PA, Hughes JM, Wells H. 1997. Dose-response associations of silica with nonmalignant respiratory disease and lung cancer mortality in the diatomaceous earth industry. ***American Journal of Epidemiology*** 145(8):680-688.

Environmental Protection Agency (EPA). 1995. SCREEN3 Model User's Guide; EPA-454/B-95-04; with Model Change Bulletins #1, #2, and #3. Officer of Air Quality Planning and Standards: Research Triangle Park, NC.

Environmental Protection Agency (EPA). 1996. Ambient levels and noncancer health effects of inhaled crystalline and amorphous silica: health issue assessment; EPA/600/R-95/115. Office of Research and Development: Washington DC.

Erren TC, Morfeld P, Glende CB, Piekarski C, Cocco P. 2011. Meta-analyses of published epidemiologic studies, 1979-2006, point to open causal questions in silica-silicosis-lung cancer research. ***La Medicina del Lavoro*** 102(4):321-325.

Faustman EM, Omenn GS. 1996. Chapter 4. Risk Assessment. In. Klaassen CD, ed. **Casarett and Doull's Toxicology: The Basic Science of Poisons, 5th ed**. McGraw-Hill: New York: NY.

Finkelstein MM. 2000. Silica, silicosis, and lung cancer: a risk assessment. ***American Journal of Industrial Medicine*** 38(1):8-18.

Frohlich E, Mercuri A, Wu S, Salar-Behzardi S. 2016. Measurements of deposition, lung surface area and lung fluid for simulation of inhaled compounds. ***Frontiers in Pharmacology*** 7:181 (10 pages).

Geenland S, Rothman KJ, Lash TL. 2008. Measures of Effect and Measures of Association. In. Rothman KJ, Greenland S, Lash TL. **Modern Epidemiology, 3rd ed**. Lippincott Williams & Wilkins: Philadelphia, PA.

Griffith J, Aldrich TE, Duncan RC. 1993. 3. Epidemiologic Research Methods. In. Aldrich TE, Griffith J. Cooke C, eds. **Environmental Epidemiology and Risk Assessment**. Van Nostrand Reinhold: Holboken, NJ.

Hardy TS, Weill H. 1995. Crystalline silica: risks and policy. ***Environmental Health Perspectives*** 103(2):152-155.

Hedges LV, Olkin I. 1985. **Statistical Methods for Meta-Analysis**. Academic Press: London, UK.

Hessel PA, Gamble JF, Nicolich M. 2003. Relationship between silicosis and smoking. ***Scandinavian Journal of Work, Environment and Health*** 29(5):329-336.

Heyder J. 2004. Deposition of inhaled particles in the human respiratory tract and consequences for regional targeting in respiratory drug delivery. ***Proceedings of the American Thoracic Society*** 1:315-320.

Huggins CW, Meyers GT. 1986. Particle size distribution of quartz and other respirable dust particles collected at metal mines, nonmetal mines, and processing plants, RI 9053. Bureau of Mines, US Department of the Interior: Washington, DC.

Hussain M, Madl P, Khan A. 2011. Lung deposition predictions of airborne particles and the emergence of contemporary disease Part-I. ***theHealth*** 2(2):51-59.

JBR Environmental Consultants, Inc. 2009(rev). Notice of Intention to Commence Large Mining Operations: Geneva Rock Products, Inc. Point of the Mountain Quarry Operations M/035/026. Utah Division of Oil, Gas and Mining: Salt Lake City, UT.

Kurniawidjaja LM. 2014. Silicosis and its progress influenced by genetic variation on TNF-alpha locus-308, TNF-alpha and IL-10 cytokine on cement factory workers in Indonesia. ***Pakistani Journal of Biological Science*** 17(3):419-423.

Lacasse Y, Martin S, Simard S, Desmeules M. 2005. Meta-analysis of silicosis and lung cancer. ***Scandinavian Journal of Work, Environment and Health*** 31(6):450-458.

Lin J, Hu GL, Fan JR, Pan D. 2009. Study on airflow and inhaled particle deposition within a realistic human upper respiratory tract. ***Journal of Physics: Conference Series*** 147:012067 (Presented at the 6th International Symposium on Measurement Techniques for Multiphase Flows).

Mannetje A, Steeland M, Attfield M, Boffetta P, Checkoway H, DeKlerk N, Koskela R. 2002. Exposure-response analysis and risk assessment for silica and silicosis mortality in a pooled analysis of six cohorts. ***Occupational and Environmental Medicine*** 59(11):723-728.

Nalder DL, Zurbenko IG. 2014. Estimating cancer latency time using a Weibull model. ***Advances in Epidemiology*** 756769.

Namati E, Thiesse J, de Ryke J, McLennan G. 2008. Alveolar dynamics during respiration. ***American Journal of Respiratory Cell and Molecular Biology*** 38:527-578.

Occupational Health and Safety Administration (OSH). 2017. OSHA's Respirable Crystalline Silica Standard for Construction: https://www.osha.gov/Publications/OSHA3681.pdf.

Pinkerton KE, Southard RJ. 2005. Silica, Crystalline. In: Wexler P, ed. **Encyclopedia of Toxicology, 2nd ed, Volume 4**. Elsevier Ltd: Oxford UK.

Poinen-Rughooputh S, Rughooputh MS, Guo Y, Rong Y, Chen W. 2016. Occupational exposure to silica dust and the risk of lung cancer: an updated meta-analysis of epidemiological studies. ***BMC Public Health*** 16:1137.

Reed WR. 2005. Significant Dust Dispersion Models for Mining Operations; Information Circular IC 9478. National Institute for Occupational Safety and Health: Pittsburgh, PA.

Richards JR, Brozell TT, Rea C, Hayden J. 2009. PM4 crystalline silica emission factors and ambient concentrations at aggregate-producing sources in California. ***Journal of the Air and Waste Management Association*** 59(11):1287-1295.

Rissler J, Gundmundsson A, Nicklasson H, Swietlicki E, Wollmer P, Londahl J. 2017. Deposition efficiency of inhaled particles (15-5000 nm related to breathing pattern and lung function: an experimental study in healthy children and adults. ***Particle and Fibre Toxicology*** 14:10 (12 pages).

Shulz H, Brand P, Heyder J. 2000. *5. Particle deposition in the respiratory tract*. In. (Gehr P, Heyder J eds.) ***Particle-Lung Interactions***. Marcel Dekker, Inc: New York, NY.

Smith AH, Lopipero PA, Barroga VR. 1995. Meta-analysis of studies of lung cancer among silicotics. ***Epidemiology*** 6(6):617-624.

Steenland K, Mannetje A, Boffetta P, Stayner L, Attfield M, Chen J, Dosemeci M, DeKlerk N, Hnizdo E, Koskela R, Checkoway H, IARC. 2001. Pooled exposure-response analysis and risk assessment for lung cancer in 10 cohorts of silica-exposed workers: an IARC multicentre study. ***Cancer Causes and Control*** 12(9):773-784.

Steenland K, Ward E. 2014. Silica: a lung carcinogen. ***CA Cancer Journal for Clinicians*** 64:63-69.

Suissa S. 1999. Relative excess risk: an alternative measure to comparative risk. ***American Journal of Epidemiology*** 150(3):279-282.

Till JE, Meyer HR. 1983. **Radiological Assessment, A textbook on environmental dose analysis.** US Nuclear Regulatory Commission: Springfield, VA.

Thomas CR, Kelley TR. 2010: A brief review of silicosis in the United States. ***Environmental Health Insights*** 4:21-26

Thun MJ, Sinks T. 2004. Understanding cancer clusters. ***CA: A Cancer Journal for Clinicians*** 54(5):273-280.

Thun MJ, Haiman CA, Linet MS, Schottenfeld D, Linet MS, Cerhan JR. 2018. **Schottenfeld and Fraumeni Cancer Epidemiology and Prevention 4th ed**. Oxford University Press: New York, NY.
Section III: The Causes of Cancer; Chapters 11-25.

Thun MJ, Hanley SJ, Travis WD. *Chapter 28: Lung Cancer*.
De Martel C, Parsonnet J. *Chapter 31: Stomach Cancer*.
Beebe-Dimmer JL, Figneau FD, Schottenfeld D. *Chapter 35: Small Intestine Cancer*.
Wu K, Keum N, Nishihara R, Giovannucci EL. *Chapter 36: Cancers of the Colon and Rectum*.
Grulich AE, Jin F, Porynten JM. *Chapter 37: Anal Cancer*.

Turner DB. 1994. **Workbook of atmospheric dispersion estimates: an introduction to dispersion modeling, 2nd ed**. Lewis Publishers: Washington, DC.

Tse LA, Yu IT, Qui H, Leung CC. 2014. Joint effects of smoking and silicosis on diseases to the lungs. ***PLOS One*** 9(8):e104494.

Vacek PM, Glenn RE, Rando RJ, Parker JE, Kanne JP, Henry DA, Meyer CA. 2018. Exposure-response relationships for silicosis and its progression in industrial sand workers. ***Scandinavian Journal of Work, Environment and Health*** (preprint) doi:10.5271/sjweh.3786.

Vilnius D, Dandoy S. 1990. A priority rating system for public health programs. ***Public Health Reports*** 105:463-470.

Yang H, Yang L, Zhang J, Chen J. 2006 Natural course of silicosis in dust-exposed workers. ***Journal of Huazhong University of Science and Technology, Medical Sciences*** 26(2):257-260.